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CASE REPORT

Multiple cervical spondylolisthesis and thoracic vertebral malformation in an 11 month-old Chilean Caballo Raza Chilena colt

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ABSTRACT. Compressive myelopathy caused by developmental cervical and thoracic malformation was diagnosed in an 11-month-old Chilean Caballo Raza Chilena colt. The patient evidenced an abnormal wide-based stance, neck muscle atrophy, upside-down neck conformation and tetra ataxia. Ataxia was exacerbated when the colt was walked down and uphill, walked with elevated head, backing and turning in tight circles. The patient showed no improvement following medical therapy, therefore, the colt was euthanised due to a poor prognosis after myelogram findings. Cervical spine malalignment (spondylolisthesis) associated with multi-level compression of the spinal cord was suspected based on cervical radiographs and myelogram findings and was confirmed postmortem. Thoracic vertebral malformation retained cartilage matrix spicules and a flare of the cranial vertebral epiphysis of the first thoracic vertebrae (T1) were also diagnosed at necropsy.

Key words: horse, cervical vertebrae, stenosis, myelogram.

Cervical vertebral malformation (CVM) (also known as cervical vertebral compressive myelopathy, wobbler syndrome and cervical stenotic myelopathy) (Reed *et al* 2007, Cardona *et al* 2013, Kühnle *et al* 2018, Szklarz *et al* 2019) is a common and widely described cause of ataxia and paresis affecting many different breeds of horses (Levine *et al* 2010). Ataxia and weakness commonly seen in horses with CVM are caused by narrowing of the cervical vertebral canal and compression of the spinal cord, often combined with malalignment and malformation of the cervical vertebrae. Two broad categories of CVM resulting in spinal cord compression have been proposed; type 1 affects young horses with compression as a result of developmental abnormalities of the cervical vertebral column, and type 2 affects older horses and typically involves a degenerative process (Nout and Reed 2003, Reed *et al* 2007).

The most important factor in the diagnosis of cervical vertebral malformation in adult horses and foals is the identification of cervical vertebral canal stenosis. The diagnosis can be made with more confidence by assessing the diameter of the vertebral canal. In many cases, cervical radiographs and myelography remain the only tools available to confirm a diagnosis of CVM and to define the site of spinal cord compression. Myelography has been considered the gold standard antemortem diagnostic test, however, sagittal diameter ratio analysis from plain radiographs may be more sensitive and specific than myelography and use of intra- and intervertebral sagittal diameter ratios may have greater predictive accuracy for diagnosis of CVM (van Biervliet *et al* 2006, Reed *et al* 2007, Hahn *et al* 2008). Although CVM is known to affect many different breeds of horses (i.e. described in 1.3% of young Thoroughbreds (Oswald *et al* 2010)) it is a condition rareley diagnosed in young and adult horses Caballo Raza Chilena and accordingly, there is no reference in the equine literature describing CVM in this breed.

The etiopathogenesis of breed predilections of CVM is still unknown but is speculated to involve genetic factors and differences in both morphometry and use. The Caballo Raza Chilena breed is characterised by a muscled short neck conformation and, furthermore, their late taming results in maintenance nutrition at younger ages avoiding overfeeding when compared to other breeds (Murúa 2006). All the above might be important factors involved in the low incidence of CVM reported in this breed. This case report describes severe cervical and thoracic malformation in an 11-month-old Caballo Raza Chilena with clinical features that, to the authors' knowledge, have not been previously described for this breed.

An 11-month-old Caballo Raza Chilena was presented to the referral hospital with progressive onset of neurologic signs, including severe ataxia of all 4 limbs. At 6-months-old, the owner noticed a ewe-necked conformation showing an upside-down neck, the development of intermittent stumbling and an abnormal stance. Clinical signs persisted until the colt was referred to the veterinary hospital. No known history of trauma was reported. Vaccination and deworming status were up to date. Diet consisted of alfalfa hay and water *ad libitum* and was deemed adequate.

At initial presentation, the colt was bright, alert, and responsive with normal vital parameters and a body condition score of 4 (range 1-9) (Henneke *et al* 1983). The colt was grade 3/5 ataxic in all 4 limbs with normal mentation and behaviour (Reed *et al* 2007). Static neurological

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Table 1. Sagittal diameter ratios and myelographic measurements for vertebral sites of the colt with cervical vertebral malformation.

Localisation	Intervertebral ratio	Intravertebral ratio	Dorsal column diameter reduction	Ventral column diameter (VB)
C2-C3	88.35%	C3 55.85%	18 %	C3= 3.49mm
C3-C4	70.81%	C4 55.50%	9.4%	C4= 3.54mm
C4-C5	74.39%	C5 53.39%	93.61%	C5= 0mm
C5-C6	76.38%	C6 58.14%	89.8%	C6= 3.65mm
C6-C7*	72.67%	C7 63.18%	-	C7= 4.25mm
C7-T1*	86.40%	T1 59.74%	-	T1= 3.78mm

*Dorsal column diameter reduction and dural diameter reduction were not obtained because the dorsal contrast column was lost at C6-C7.

exam revealed abnormal (base-wide) limb posture, neck muscle atrophy (cervical serratus, rhomboid, and splenius muscles), ventral displacement of the cervical vertebrae and trachea, and low head carriage (figure 1A). No cranial nerve deficits were observed. Gait analysis showed a severe lack of coordination of motor movements in all four limbs. Ataxia was exacerbated when the colt was walked down and uphill, walked with the head elevated, backing, and turning in tight circles. The colt also showed signs of brachial plexus compression during certain manoeuvres, became stiff in the front limbs, and almost dropped. Dynamic tail pull showed a lack of resistance, and the hind limbs were weak and easily pulled off balance followed by the colt taking several strides to recover.

The colt was treated with flunixin meglumine[1] (1 mg/kg bwt i.v b.i.d.), dexamethasone[2] (0.01 mg/kg bwt i.v s.i.d.), dimethylsulfoxide 99%[3] (100 mL in 1 L Lactated Ringer's solution[4]) i.v. s.i.d., during 4 days. Despite treatment, no improvement was observed, and the colt was referred to the veterinary hospital for further diagnostics including imaging.

Complete blood count and serum biochemistry profile were unremarkable. Standing cervical radiographs showed severe malalignment (ventral subluxation) of the cervical vertebral column between C4-C7, but no narrowing of the vertebral canal was noted (figure1B, table 1). A myelogram was subsequently performed under intravenous anaesthesia in a padded recovery room as described by Grant and Paterson (2006) (figure 2). An 18G spinal needle was placed in the atlantooccipital space, 40 mL of cerebrospinal fluid (CSF) were withdrawn, and 40 mL of contrast agent (iodixanol [Visipaque 300][5] 300mg/mL) were injected intrathecally. The myelogram revealed spinal cord compression in neutral, flexed, and extended radiographs at C5 (loss of ventral dye column within the vertebral body), between C5-C6 (thin dorsal column), and between C6-C7 (loss of dorsal dye column) (table 1). Contrast material was observed until caudal T1 (figure 3). No thoracic abnormalities of bone were presumed from plain radiographs nor myelogram. Based on these findings, developmental cervical vertebral malformation/malarticulation (spondylolisthesis) was diagnosed.

The colt recovered uneventfully from the myelogram. However, due to the myelographic findings of vertebral canal stenosis, multiple areas of spinal cord compression, and associated poor prognosis, euthanasia and postmortem evaluation were elected. At necropsy, malformation of the dorsal lamina of C5 and C7 were observed causing stenosis of the vertebral canal. Additionally, thoracic vertebral malformation associated with retained cartilage matrix spicules and a flare of the cranial vertebral epiphysis of the first thoracic vertebrae (T1) were also diagnosed at necropsy. Spinal cord histopathology was not performed, and no other organs were examined postmortem.

Compressive stenotic myelopathy is the most common non-infectious cause of spinal ataxia in young and adult horses (Levine *et al* 2010, Janes *et al* 2015). The first type of CVM occurs in young horses and is essentially developmental in which malformation/malarticulation of the cervical vertebral column causes spinal cord compression (van Biervliet *et al* 2006). Compressive vertebral malformation in young horses (<3 years old) is a multi-factorial disease affected by genetic predisposition and environmental influence such as gender, nutrition, hormonal changes, exercise, trauma, and rate of growth (van Biervliet *et al* 2006, Levine *et al* 2008), with Thoroughbreds and Warmbloods being overrepresented (Levine *et al* 2010, Piercy 2011). Different types of lesions have been reported in young horses (osteochondrosis, osseous cyst-like structures, fibrous tissue replacement of trabecular bone, retained cartilage matrix spicules, and osteosclerosis) providing evidence that developmental abnormalities during cervical vertebrae growth and

[1] Febrectal™, Dragpharma, Santiago, Chile.

[2] Hasyun™, Dragpharma, Santiago, Chile.

[3] DMSO 99%, Valhoma, Tulsa, OK.

[4] Ringer-Lactato, Baxter Chile, Santiago, Chile.

[5] Visipaque™ 300, GE Healthcare Inc. Chile, Princeton, NJ.

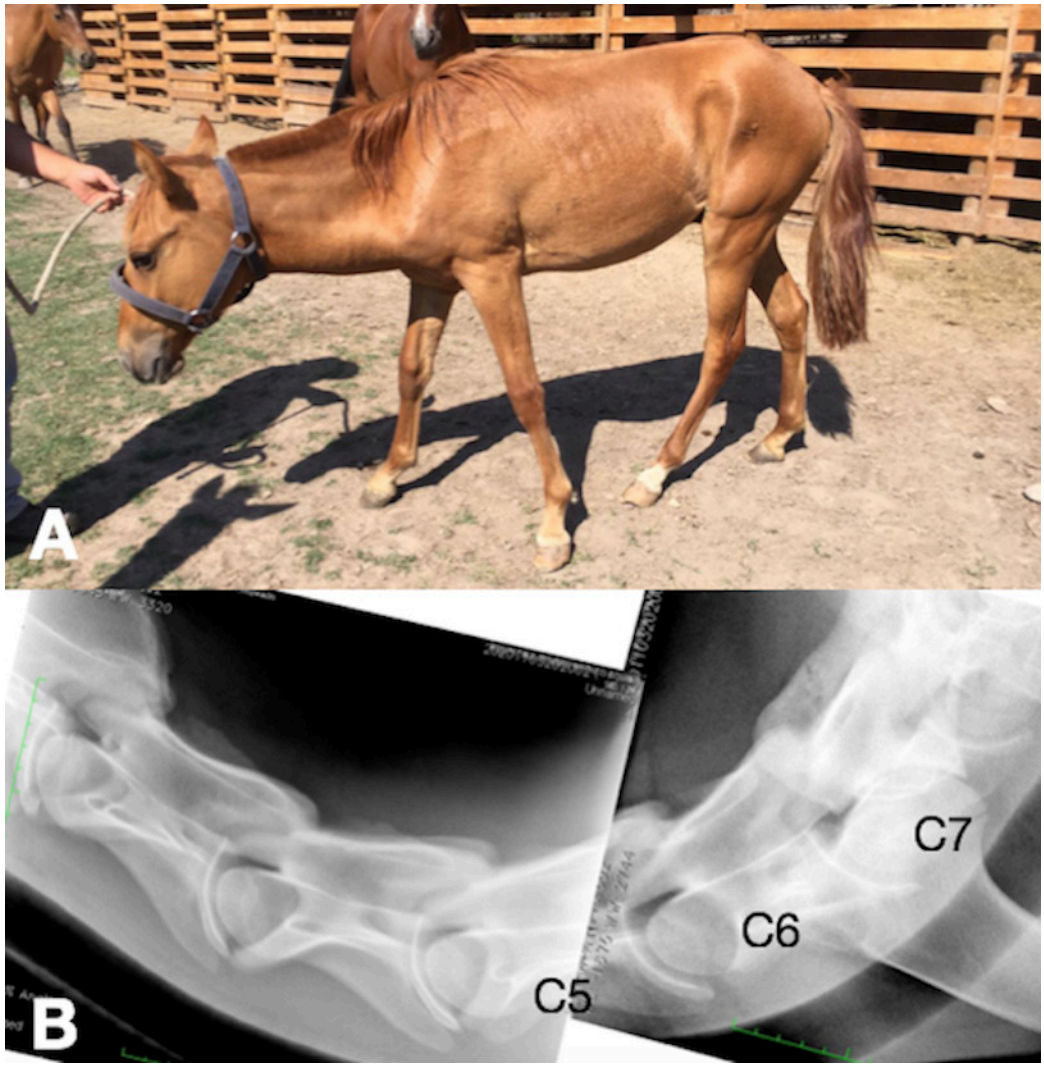

Figure 1. A) Photograph of the colt presenting a notorious alteration at mid and lower neck with low head carriage, abnormal stance in the fore and hind legs and muscle atrophy of the neck. B) Reconstruction of the colt cervical radiographs in neutral position showing malalignment (spondylolisthesis) most evident between C4-C5 and C6-C7 without apparent intervertebral space abnormalities.

maturation are important in their pathogenesis (Stewart *et al* 1991, Janes *et al* 2015, Bergmann *et al* 2020). Also, anomalous C6 with absence (symmetric or asymmetric) of the ventral lamina of the transverse process might be associated with developmental spinal stenosis (DeRouen *et al* 2016). The second type of CVM is most often seen in mature horses and has been commonly characterised by malformation with degenerative joint disease of the articular processes, wedging of the vertebral canal, periarticular proliferation with or without a synovial or epidural cyst and overt fractures of the articular processes (Reed *et al* 2007). Spinal cord compression may also result from traumatic injury (Matthews and Nout 2004, Denoix 2005), vertebral body fracture (Matthews and Nout 2004), vertebral neoplasia (Hirsch *et al* 2009), discospondylitis (Furr *et al* 1991, Denoix 2005), intervertebral disk protrusion (Nixon *et al* 1984), epidural hematoma (Cunha dos Santos *et al* 2014, MacMillan *et al* 2020), ischemic fibrocartilaginous embolism (Sebastian and Giles 2004, Dörner *et al* 2015) and arachnoid diverticulum (Allison and Moeller 2000). Furthermore, spinal cord compression may be also associated to congenital malformations such as hemivertebrae (Wong *et al* 2005), butterfly vertebrae (Rendle *et al* 2008), block vertebrae (Perris *et al* 1994), occipitoatlantoaxial malformation (Mayhew *et al* 1978, Watson and Mayhew 1986), atlantoaxial subluxation (Witte *et al* 2005) and atlantoaxial instability (Rush 2012, Cole *et al* 2017). However, congenital anomalies of the vertebral column are reported infrequently. In Chile, as elsewhere, CVM is a disease commonly diagnosed in young and adult Thoroughbreds and Warmbloods. Despite being the most prevalent cause of spinal ataxia in the abovementioned breeds, CVM is rarely noted in young and adult Chilean horses and thus has not been described in the literature in

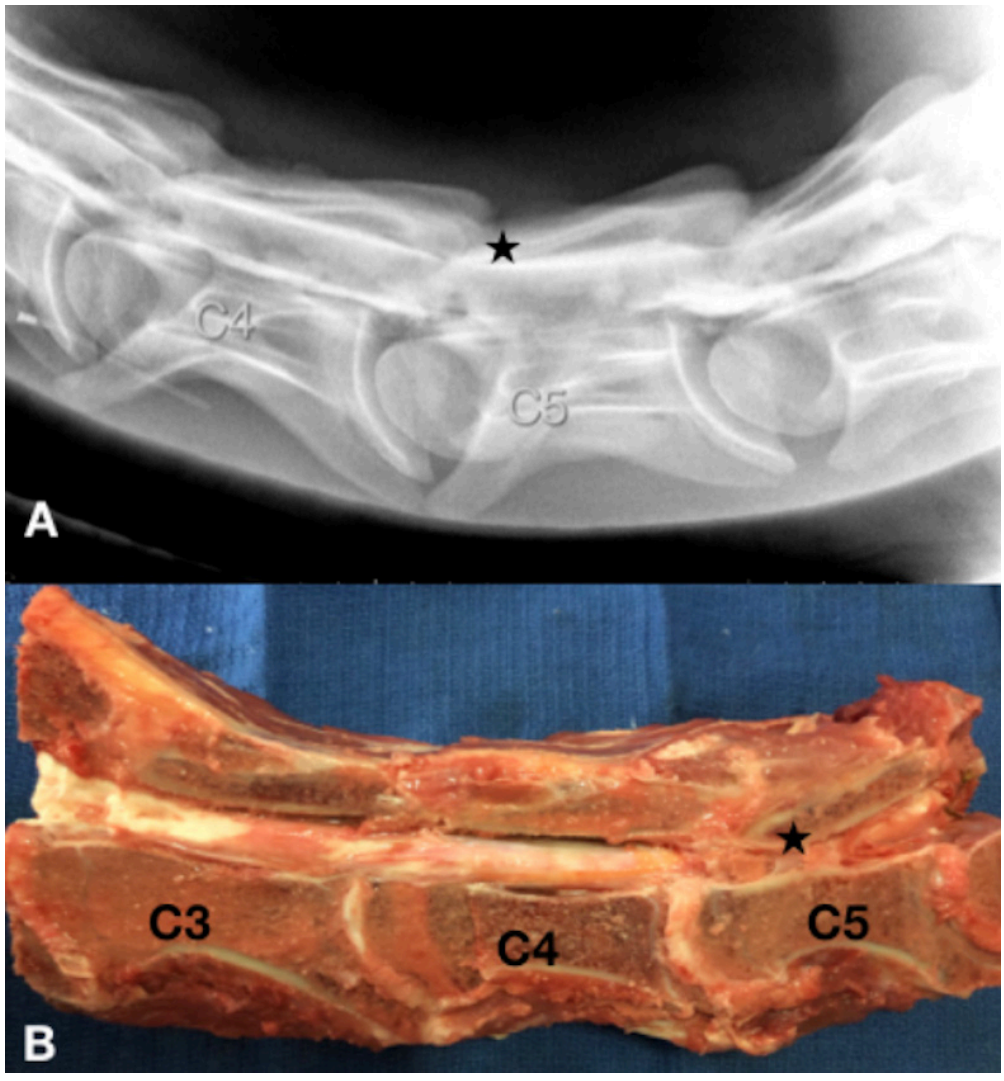

Figure 2. A) Contrasted cervical radiographs with neck in neutral position showing complete loss of the ventral dye column at C5. B) Anatomical dissection with longitudinal cross section of the C3-C5 cervical spine showing the site of spinal cord compression at C5 (black star).

this breed until today. The Caballo Raza Chilena horses are characterised by a lower growth rate and a muscled short neck conformation when compared to other breeds. It has been previously described that both rapid growth rates and length of the neck could be influencing the development of CVM (Rooney 1969, Levine *et al* 2008). Also, late taming and the start of training at approximately the age of 3 for this breed (Murúa 2006) results in a maintenance diet without overnutrition. All these factors could be involved in the low incidence of CVM in this breed.

In this case, the colt manifested classic signs of cervical spine impingement associated with ventral subluxation, also referred to as spondylolisthesis (Denoix *et al* 2005, Denoix 2007, Dyson *et al* 2020) between C4-C7. Also, a bony formation in the dorsal laminae of T1, probably as a result of retained cartilage matrix spicules (Janes *et al* 2015), along with a flare of the cranial vertebral epiphysis of T1 was noticed postmortem. Spondylolisthesis refers to a displacement of one vertebra on another (Denoix 2007) and it has been identified with transrectal ultrasonographic examination at the lumbosacral junction in adult horses (Denoix *et al* 2005), in the atlantoaxial joint in foals (Witte *et al* 2005) and recently in the cervical and cranial thoracic vertebrae associated with intervertebral disk disease in adult horses (Dyson *et al* 2020). Although the attenuation of dye at T1 during myelogram was attributed to the typical loss of dye column at that level, it is possible that these malformations and associated suspected impingement impeded the dye from progressing farther caudally beyond T1.

We were not able to delineate a specific cause for the cervical and thoracic malformations encountered in this horse. Abnormal *in-utero* development or position during pregnancy might have played a role in the development

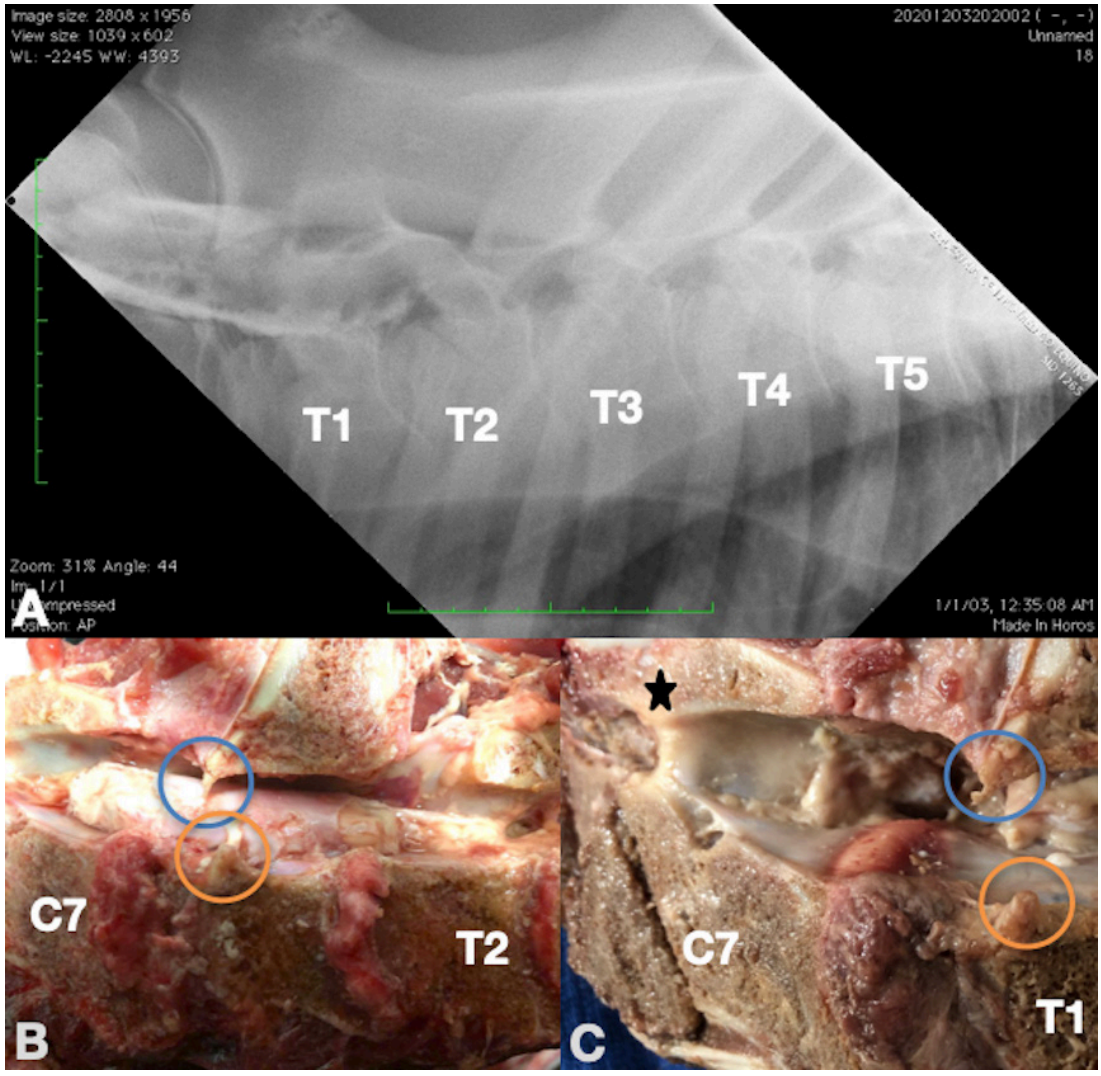

Figure 3. A) Radiograph taken during myelogram of proximal thoracic vertebrae (T1-T5). Contrast material is only observed until caudal T1. No thoracic abnormalities of bone were presumed from plain radiographs nor myelogram. B) Anatomical dissection with longitudinal cross section of the C7-T2 spine showing two large spicules, one in the cranial dorsal laminae of T1 (blue circle) and one in the cranial vertebral body of T1 (orange circle). C) Photograph showing spinal canal narrowing at C7 (black star) representing a second site of spinal cord compression.

of this condition, but neurologic deficits would have been expected to occur much earlier in the horse's life and no abnormalities were detected neither by the owner nor the field veterinarian before weaning. On the other hand, even though the diet was deemed adequate for this colt, it is not known to what extent trace nutrients, such as copper and zinc deficiencies, or calcium and phosphorus imbalance may have contributed to the presentation of the malformation observed in this horse. To our knowledge, there are no known calcium, phosphorus, copper, or zinc deficiencies in the area where the colt lived (Maldonado 2006). On the other hand, and even though blood and soil selenium concentrations were not measured, the south of Chile is characterised for being deficient in selenium (Crempien 1988, Tapia 2013) and as it is widely described that selenium is an essential microelement for animal development (Zarczyñska *et al* 2013, Hung Son and Duong Huyen 2019). However, similarly aged colts on the property, as well as direct relatives of the colt, were free from abnormalities. Despite the above, some degree of nutrient abnormality in the colt's diet cannot be ruled out nor can an underlying error of metabolism.

Radiographic indicators and measurements of the cervical canal height categorised by standard minimal sagittal diameter, and intravertebral and intervertebral ratios have been widely used despite the potential for false positive and false negative determinations of canal stenosis (Janes *et al* 2013). According to the above reference, spinal cord compression is possible if intra and intervertebral ratios are >50% for C4, C5 and C6 or >52% for C7 (Reed *et al* 2007), as shown in the colt presented in this case (table 1) in which the ratios were within normal parameters, but

spinal cord compression was only evident via myelography. For this reason, diagnosing sites of compression may be more accurate when both sagittal diameter ratios and myelographic measurements are used together (Hahn *et al* 2008). Additionally, postmortem magnetic resonance studies have shown that vertebral canal area and cord canal area ratios are better parameters to predict the location of cervical canal stenosis than only the sagittal plane of canal height (Janes *et al* 2013).

An important aspect that should be considered from this report is that radiographs and myelography can certainly underestimate the severity of spinal cord compression (Gough *et al* 2020). There are still some abnormalities that cannot be definitively diagnosed with conventional nor contrasted radiographs. Computed tomography (CT), or magnetic resonance are the imaging modalities that can appraise more accurately sites of spinal cord compression, vertebral structures, or bone changes and can help to confirm a specific diagnosis (Janes *et al* 2015, Gough *et al* 2020). Unfortunately, these advanced imaging tools are not available in many practices. Therefore, intra- and intervertebral sagittal ratios, dural diameter and contrast column reduction measurements often remain the only tools available to diagnose, prognose and propose treatment despite the risk of false positives or false negatives (Janes *et al* 2013).

Neurological clinical signs associated with cervical spinal cord compression were very characteristic in this case and considering the exacerbation of the signs with specific maneouvres, dynamic compression was presumed following clinical examination. However, the myelogram did not show significant differences in dye column reduction between changes in neck positioning (neutral, flexed or extended). Although no dynamic compression was evident during the myelogram, attenuation, and loss of contrast columns within vertebral bodies were observed in multiple sites, hence a static compression at C5, C6 and C7 was deemed more likely. Furthermore, even though the spinal cord compression was diagnosed after the myelogram, the severity of compression was only evidenced after necropsy (figures 2 and 3). Additionally, necropsy revealed a significant spicule in the cranial aspect of the dorsal laminae of T1 along with a flare of the cranial vertebral body of T1, neither of which were visible in the radiographs (figure 3). Most cases of thoracolumbar malformations described in horses have been manifested as gross deformity, usually without associated spinal cord compression and ataxia, and they include mainly transitional abnormalities and vertebral axis deviations (Lerner and Riley 1978, Denoix 2005). Thoracic vertebrae malformation causing neurological signs has been described only in a few horses before (Johnson *et al* 1997, Rush 2012). Even though thoracic spinal compression was not initially suspected after the myelogram, after correlating the clinical signs and necropsy findings, it is possible that the attenuation of dye at T1 represented compression at that level. Spinal cord histopathology would have further characterised the extent of compression but unfortunately, was not performed.

Although surgical treatments have been reported (Nixon 1991, Moore *et al* 1993, Kühnle *et al* 2018, Pezzanite *et al* 2021), further procedures were foregone in this case, and euthanasia was selected due to imaging findings, severity of clinical signs and associated poor prognosis.

Cervical malformation with spinal cord compression and ataxia is a common cause of neurological deficits in young horses of various breeds, while neurologic signs associated with thoracic vertebral malformation have been rarely reported. To our knowledge, this is the first reported case of multiple cervical spondylolisthesis and thoracic vertebral malformation with associated multi-level cord compression in the Caballo Raza Chilena horse. Vertebral malformation and malalignment should be considered as differentials for ataxia and neurologic deficits in young horses, including this Chilean breed, even if not evident radiographically.

ETHICAL STATEMENT

The owner agreed to the presentation and divulgation of this case report.

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